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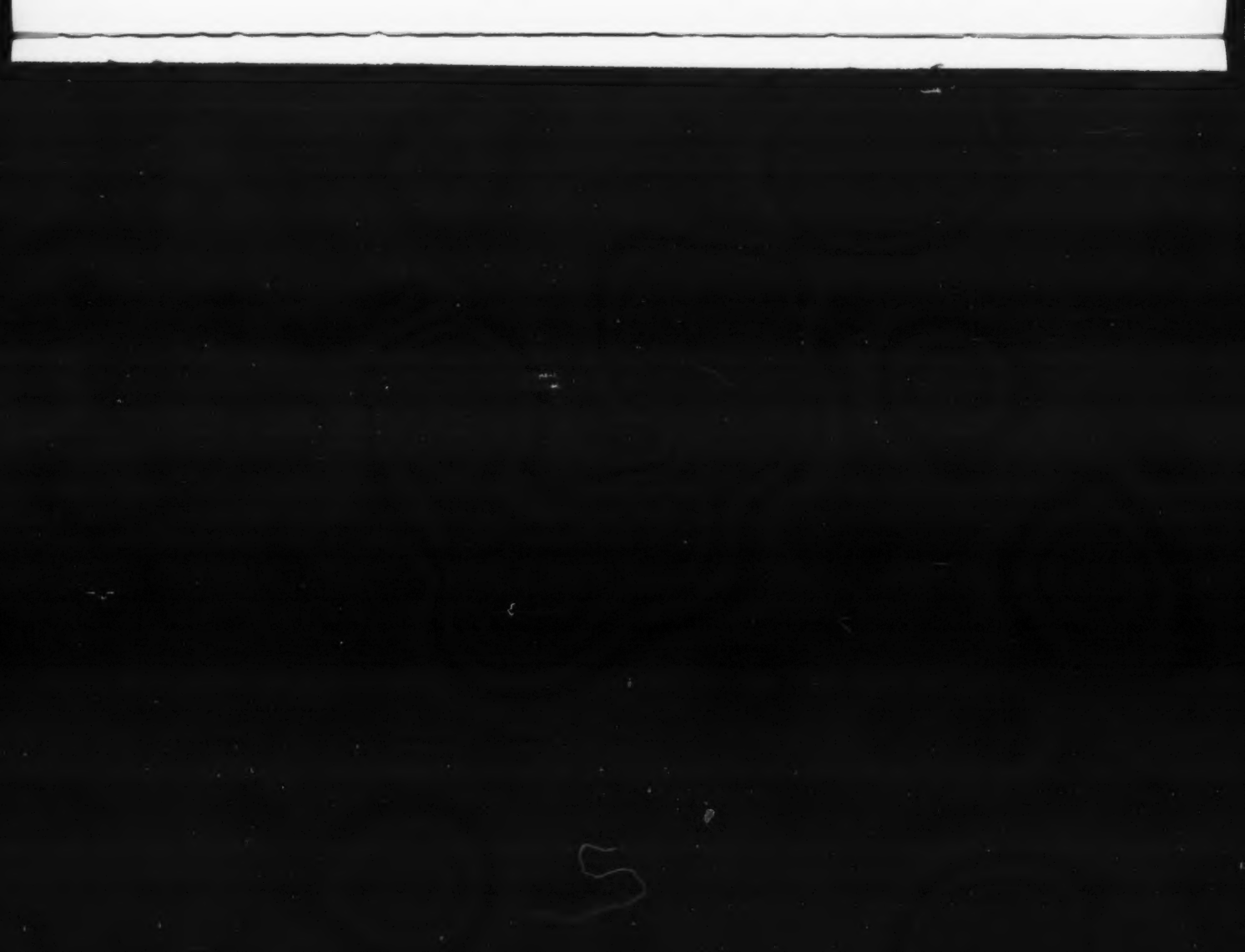

SPECIAL REPORT

THE RÔLE OF COW'S MILK IN GASTRO-INTESTINAL ALLERGY OF CHILDREN

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The substances produced in the allergic individual resemble histamine and cause symptoms resembling those due to stimulation of parasympathetic nerve endings, depending upon the location of the shock organ; i.e., the nose, bronchi, gastro-intestinal tract or the skin.

It is difficult to rationalize the variety of symptoms, referred to the gastro-intestinal tract, particularly in cases of constipation and of diarrhea. Bockus[1] states, "Ordinary stimulation of parasympathetic innervation of the intestines causes tonus and peristalsis, whereas splanchnic nerve stimulation tends to diminish tone and inhibit movement. The response to stimuli, however, is not consistent and may be reversible, possibly the result of a mixture of cholinergic and adrenergic fibers in both nerves."

It has been postulated that the basic condition of allergy is due to an imbalance in the autonomic nervous system. Many allergic individuals have vagotonia as evidenced by sweating, slow pulse and low blood pressure; others have sympathicotonia. It is possible, therefore that in individuals with increased vagus tone, gastro-intestinal allergy may result in diarrhea, while in patients with sympathicotonia, constipation occurs.

Feinberg[2] states that the importance of gastro-intestinal allergy is not fully recognized because many functional and organic diseases present symptoms strikingly identical to those of allergy, and furthermore there is a low incidence of positive skin reactions. The value of skin tests with foods is debatable. The extracts used for testing are made from basic foods. It is a reasonable deduction that a basic food in its ingested form is a different protein molecule from that which is absorbed after it has been changed into an entirely different chemical substance by digestive enzymes. Furthermore, some food extracts contain irritating factors which may cause a false positive reaction. When a positive skin test is produced with a testing solution, the skin is acting as the shock organ. However, for a food to cause gastro-intestinal symptoms, the shock organ must be the gastro-intestinal tract.

The late Dr. Vaughn of Richmond found that 60–90 minutes after the ingestion of a food to which a patient is hypersensitive, the white blood cells of the peripheral circulation were decreased by one thousand or more. Because of the large number of foods, Vaughn's leukopenic index has limited possibilities.

Thus, if skin or other tests are performed they should not be used as the only evidence of the allergic complaint. However, they may be used as a starting point in the food elimination diets.[3]

The gastro-intestinal allergic syndrome has received rather scant attention by the Pediatrician and Internist. Current periodicals seldom carry observations on this subject. Pediatric texts devote a few paragraphs to the problem. This paper presents clinical data which we believe of importance in a large group of children coming under observation in private practice. These children present a variety of symptoms simulating organic disease in many instances. And while the presenting complaints may be dissimilar, there is a similarity of basic patterns. The patterns have their origin usually in infancy with changes in clinical manifestations as growth and development take place.

Changes in symptoms are probably the result of increasing sensitivity to new foods and/or increasing desensitization to original offending proteins. These changes are based on the well known fact that allergic children with respiratory symptoms must be retested at intervals to inhalants since their sensitivity to these substances change with age. Many of these children show evidence of allergy to food for the first time at the age of three years or later. This follows the pattern seen in respiratory allergy where the first attack occurs in adolescence or adulthood. There is no adequate explanation for this late activity of the allergic state except that constant association with the allergen finally completes the sensitization process.

On the other hand it is claimed by McGee[3] that hiccough in the fetus is an allergic phenomenon. Our earliest experience occurred in a two months old breast fed infant who reacted to the first feeding of cow's milk by shock reactions of vomiting, cyanosis, dyspnea and sweating. He was slowly desensitized to milk and enjoyed excellent health until he later manifested constipation, anorexia, pallor, lassitude and irritability at the age of eight years; these symptoms were promptly relieved by again omitting milk from the diet.

A thorough history is most important for properly evaluating the presenting complaint. This is one instance in which the mother must be allowed ample time to unburden herself of the numerous symptoms present and past. The history of necessity will consume 75% of the time allotted and this point cannot be overemphasized. It is possible, by close questioning, to obtain a family history of allergy in 70–80% of these patients. This is obviously important. Reactions to earliest feeding must be detailed. Various changes in formulae and the infant's reaction thereto must be noted; on occasion a malt sugar has been found to be the offender. Following this occurs the addition of fruit juices or Vitamin C substitutes and Cod Liver Oil. Usually the order in which other foods are added to

the diet may be set down as cereals, vegetables, egg, meat, other starches, stewed fruits, various baby mixtures, etc. As these different foods are added, changes in symptoms may become manifest.

For the sake of clarity we have separated our patients into arbitrary groups headed by the significant complaint. Interestingly these groups embrace a more or less well defined age period. It must be remembered, however, that symptoms merge one into the other as growth and development take place and new foods are added to the diet. Development of mental awareness to discomfort materially modifies symptoms in many children.

COLIC

Vomiting, distention, diarrhea, excessive crying and constipation are exhibited during the first few weeks of infancy. While the great majority of these patients are in artificially fed infants, a few occur in the breast fed. It has been shown that foreign protein may be passed through the mother's milk to the infant. The history of colic is of short duration but involves such significant data as hunger satisfaction, sucking satisfaction, physical comfort, feeding technique such as too small nipple holes, dogmatic schedules, etc. It is recognized that hunger accounts for a large percentage of crying in these infants.

Organic lesions and infections should be looked for in all instances with the above syndrome. Of these, otitis media and pyuria are the most commonly encountered.

When these factors are eliminated, sensitivity to an offending protein intake must be considered. Prolonged boiling of cow's milk may be sufficient, elimination of a malt sugar or a change to goat's milk may answer the problem. Should these fail, a substitute for milk may be made, using a soy bean or other hypo-allergic preparation. One excellent substitute was found to be fortified with yeast—a very definite offender in a certain number of highly sensitive infants. It is to be deplored that there are so few simple foods on the market for infant feeding which have remained free of "vitamin contamination." This is vividly illustrated in the case of C. M. who had survived several months of 24 hour "colic" which but slowly diminished in severity. Even at the age of 18 months she had never awakened less than three times a night. She was constipated, pallic (though not anemic), had an erratic appetite, was irritable and otherwise disagreeable. There was a family history of allergy. She was placed on a well known milk substitute and while greatly improved continued her disagreeable behavior. This same preparation was obtained without yeast and almost immediately she slept throughout the night and in two months was a model child. A trial of the year containing preparation was again made but the same symp-

toms returned. She has remained symptom-free for the past year on a milk and yeast free diet.

Case histories of "colic" might be cited ad nauseum all of which can be duplicated by anyone engaged in infant feeding. Foods other than milk are guilty of causing distress. Orange juice is vomited frequently and also causes pain; the same is true of preparations of cod liver oil. It is worthy of note that the irradiation of milk by ultra violet light was the cause of unknown changes in milk, with resulting "colic" symptoms in many infants.

Later in the life of allergic infants and those with latent sensitivity, is the group which we have included under the following heading.

CONSTIPATION AND DIARRHEA

These patients simulate the asthmatic. Constipation in the allergic child is constant and changes only by the use of laxatives, enemas, etc. except when interrupted by diarrhea. The stools are large, hard, dry, crumbly, grayish-white and may not even soil the diaper. The stools have a rather foul odor and when diarrhea occurs, the odor is increased. Diarrhea usually occurs periodically in the course of constipation, but on occasion has been seen from infancy. Two cases will illustrate this.

Case # 1: G. C., a white male of four years. The mother gave a history of his having very large, mushy, foul stools—three to six in number—from early infancy. He was never nursed. Many formula changes were made with various types of cow's milk with the inclusion of casein, cereals, etc. None of these changed the character of the stools. Despite this intestinal condition his general nutrition had continued to be excellent with normal developmental progress. There were no abnormal reactions to accessory vitamins or solid foods. Milk was removed from his diet with immediate improvement to normal stools within the week. He remained off milk for four years, continuing to grow and develop into a normal robust boy.

Case # 2: D. M. was seen several months ago with a similar history. He is now eight years of age. During his lifetime he had never less than 6 to 8 stools daily. He had one or more stools each night. A pot had to be carried in the car whenever he accompanied the parents on a trip. He is a tall, thin, muscular boy without evident malnutrition. He tires rather easily, however, and consequently does little in an athletic way. Milk has been removed from his diet and for six months he has had normal stools. Ice cream has ceased to have an appeal for him.

In neither of the above patients was pain and gas a striking feature. A family history of allergy was present in each.

Constipation in infants and children has always been a field for the use of innumerable remedies. Laxative advertisements adorn the pages of mag-

zines and newspapers and jam our radios. Numerous articles appear in medical journals giving scientific advice on balanced diets, roughage, exercise and massage ad lib. None have appeared so far as we know in which a food was blamed as the cause of constipation. And yet there is an old folk axiom that "Cow's milk is constipating." In any group of children under usual feeding regimes, there are a number who persistently eat an unbalanced diet, but do not suffer from constipation. On the other hand a part of this group enjoys an ideally balanced diet but have elimination only when given laxatives or enemas. A goodly number of this latter group have psychic causes for constipation and many are examples of poor toilet education. There remains a large proportion which we believe have specific food sensitivities causing hard, dry stools associated with spasm of the colon. The empty colon can be felt as a large firm cord in the left lower quadrant. This can be demonstrated by fluoroscope when the offending substance is given with barium.(1) It is our opinion that constipation is produced by the ingestion of certain foods, more often than the result of a so-called unbalanced diet.

A case in point of J. S., who was first seen at the age of three years. Constipation was a serious problem from early infancy. His mother had followed to the letter the advice and suggestions of her pediatrician. Various changes were made in the formulas given during infancy. Special attention was given to balancing solid foods with emphasis on fruits, vegetables, vitamins, etc. Toilet habits were corrected as indicated. Milk was eliminated from the diet and soft stools were passed daily without undue effort after an interval of a few days. The mother tried on three occasions to replace milk but met with the same problem of hard, dry stools which were passed at intervals of 2–4 days with the aid of enemas. This boy is now nine years old, has a perfect dental record and has never shown any ill effects of absence of milk from the diet.

Thus far we have discussed problems considered to be due to food allergy producing symptoms of a very distinctive nature. These are obvious to the senses of hearing, sight and smell. Perhaps the largest group of these food disturbances can be designated as follows.

NUTRITIONAL

This term includes underweight, anorexia, poor weight gain, pallor, lassitude, fatigue, poor school progress, irritability, constipation, foul breath, frequent colds, abdominal pain or distress, nausea or vomiting, bed wetting, and periodic bouts of temperature. Many of these children present one or more of these symptoms while others run the whole list over a period of time. Obviously such a complaint as underweight and poor weight gain show physically when measured and evaluated by reasonable standards. Pallor is fre-

quently not due to anemia since most of these children have been flooded with all sorts of tonics, a majority of them containing iron. Lassitude and fatigue are associated with poor tissue turgor, a large abdomen with atonic musculature and evidence of intestinal distention. Poor school progress is associated with slow sluggish response to questioning and poor musculature as well. Constipation and foul breath are accompanied by coated tongue (frequently geographic), distended abdomen, and hard fecal masses in the left lower quadrant. When such a child is seen at the time of high temperature he may or may not have vomited or been nauseated. The throat may be slightly reddened and tongue coated. Prostration and apathy are out of proportion to the local throat infection. The temperature promptly subsides with a cleansing enema and the distended abdomen disappears. These bouts of temperature and the frequently recurring upper respiratory infections, as well as nausea and vomiting, have a very definite cyclic pattern, varying in the individual from two weeks to four month intervals. A proportion of this group have a significant eosinophilia in the blood. The group with frequently recurring colds may show an excess of eosinophilic granules in the nasal mucus. These latter usually have rather persistently large, pale inferior turbinates and enlarged tonsils even during a quiescent phase. As pointed out above, skin tests are of little value in the majority of patients. When positive reactions can be obtained in a complicated problem, they are of value and should be done on occasion. The history is the most important means of arriving at a workable diagnosis. This must be detailed so as to follow every step in the development of the presenting syndrome including changes in food additions, changes in management, changes in household personnel or in schools, or the arrival of a sibling, etc. Psychic disturbances must not be overlooked, nor should a child be blamed for irritability caused by some article of diet deemed necessary to his welfare. Organic causes for many of these symptoms are of course possible. A good physical examination and laboratory data may be needed. It is assumed that these aids will be used to the best advantage. Furthermore vitamin deficiencies must be kept in mind and the dietary evaluated accordingly. The group of patients finally corraled as food sensitive will respond dramatically when the offender is removed from the diet. The following patients will illustrate one or more complaints as enumerated above.

Case #1: P. D. was first seen at eighteen months of age with chief complaints of high temperature and distended abdomen. These were relieved by enema with resulting large, grayish white, foul stool. This was one of four or five previous attacks. The patient was pallid, had a chronically distended abdomen and poor tissue turgor. There was a history of colic in infancy with constipation and anorexia. Attempts were made to modify diet and reduce milk intake. The above pattern continued until milk was completely omitted. Improvement followed but good behavior

was not obtained until citrus fruits and eggs were eliminated. Physical condition improved as evidenced by less abdominal distention, better color and tissue turgor. Bouts of high temperature with abdominal distention occurred infrequently. Attempts were made on occasion to replace milk beginning with very small quantities. Invariably constipation, restlessness and anorexia followed in a week or ten days. And during this period of three years there were more attacks of upper respiratory infections than should normally occur in a child of this age. During the past one and one-half years no attempt has been made to replace milk and the upper respiratory infections have been notably less in number. Though greatly improved by a milk free diet, we do not believe that we have completely eliminated all reacting foods.

Case #2: J. F., an eight year old male, was first seen with complaints of pallor, lack of weight gain, anorexia, frequent upper respiratory infections, poor school progress (partly due to being out of school more than half the time), coated tongue, foul breath and abdominal distress. He was weaned because of vomiting and colic. Worse reactions followed with various cow's milk formulas until solids were added. There was some subsidence of symptoms but constipation and poor appetite continued. Suppositories, tonics and laxatives were included in the family budget along with good foods. The mother drank large quantities of milk during pregnancy, supposedly for the infant's welfare. Removal of milk from the boy's diet reversed completely his behavior pattern. Incidentally, the tonsils and adenoids had been removed several years previous to his coming under our observation.

Case #3: G. H., a 12 year old boy, had the usual history of infant feeding problems including a distressing lack of physical development. Pertussis caused marked vomiting of milk which was removed from his diet and weight gain occurred to the amazement of everyone but without their recognizing the significant cause. Attacks of asthmatic-type wheezing with colds were frequent. Abdominal pain became a frequent complaint. Finally a normal appendix was removed despite an 18% eosinophilic blood finding. The abdominal pain continued until milk was eliminated.

Case #4: C. J. represents the counterpart of seasonal hay fever in that this infant from the age of two months produced an enormous excess of nasopharyngeal mucus. This became so excessive at times that the mother became alarmed lest the infant asphyxiate. It was necessary to use postural, in fact an upside down position, in order to clear the pharynx. A soy bean milk substitute completely cleared this problem.

Case #5: B. T. was seen June 25, 1944 at the age of three years with the chief complaints of loss of weight following a recent upper respiratory infection, intermittent attacks of diarrhea with mucus stools and constipation since one year of age. During the past two months frequency and burning

on urination were also present. Physical examination was negative except for pallor, large tonsils and a protuberant abdomen. Stool examination was negative except for considerable macroscopic and microscopic mucus. She was tested with foods and reacted to cow's milk, pork, orange, spinach, banana and pineapple. These foods were eliminated from her diet. The stool was examined ten days later and only microscopic mucus was found. Two months later evaporated milk was tried on cereal with resulting diarrhea. Oral desensitization to milk was started. She is now able to take 24 ounces daily. Two months later oral desensitization to orange was carried out. Gradually other foods have been added and there has been no return of symptoms. Apparently desensitization to milk has been accomplished in this instance at least temporarily.

COMMENT

An attempt has been made to correlate the symptom complex of abnormal gastro-intestinal reactions to foods with respiratory reactions to inhalants. Both manifest discomfort of pain, spasm, excess mucus production, eosinophilia and shock reactions. Absence of positive skin tests to suspected foods does not eliminate the clinical diagnosis of "gastro-intestinal allergy."

A thorough history is the most important part of the examination and should include careful investigation of family allergy.

When these patients are first seen in later childhood, constipation, malnutrition, anorexia, fatigue, frequently recurring upper respiratory infections are the common presenting complaints. A history of infantile colic or constipation is invariably present. It is interesting that very few of these patients give a history of eczema. Infantile eczema is more apt to precede asthma of later life.

Milk has been found to be the most common offending food. The removal of milk and/or other reacting foods is followed by prompt relief of symptoms. Many of these patients can be desensitized to milk by oral administration, which desensitization may or may not be permanent.

The absence of milk in the diet does not, in our experience, lower the blood calcium or phosphorous. Neither has it, in cases observed over a period of years, resulted in increased dental caries.

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ACUTE INFANTILE HEMIPLEGIA OF VASCULAR ORIGIN

Case Report No. 111

R. O. Warthen, M.D. C. W. 47-11747

C. W., a 3 year 4 month old colored male was admitted to Children's Hospital on November 7, 1947 at 3 a.m. in a comatose state. He had been in apparent good health until two hours prior to admission at which time he was found in a generalized tonic and clonic convulsion with his head turned to the right, eyes rolled up, jaw clamped shut, upper extremities flexed and lower extremities extended and stiff. Rapid respirations accompanied this convulsive seizure. This seizure persisted until admission to the dispensary two hours later when he received a hypodermic of one grain of phenobarbital and lapsed into a state of deep sleep.

Physical examination on admission to the ward revealed a well developed, well nourished 3½ year old colored male in a post convulsive state with moderate trismus, flaccid extremities, slow irregular respirations and nonresponsive to external stimuli. Temperature was 100.2°, pulse 100 and respirations 12. The eyes were in mid-dilatation and reacted to light. The neck was not stiff and Kernig and Brudzinski's signs were negative. Slight cervical and inguinal lymph gland enlargements were noted. Numerous café-au-lait spots were present on the skin.

Neurological examination, in so far as one could be performed in this state, revealed an absence of all deep and superficial reflexes, absent Babinski's bilaterally, hypotonia of all extremities and hypertonia of the jaw muscles.

The child's difficulties apparently dated back to the age of nine months at which time had had tonsillitis and a high fever accompanied by a generalized convulsion lasting two to three minutes. Following this initial seizure he was noted to drag his left leg slightly when walking and since the seizure he had not used his left arm as much as would be expected of a normal child. This mild hemiparesis had persisted to the present admission. Again at the age of 2½ years he experienced another generalized convulsion lasting approximately 30 minutes and similar to the previous one at nine months of age. No change in his hemiparesis was noted following this second seizure.

The child had been somewhat retarded mentally since birth and prior to 4 months before admission spoke no understandable words. During the 4 months prior to admission he had spoken single words such as "mama," "dada," "water," etc. and on admission had a vocabulary of about 5 to 6 words. His physical development was normal for his age. He sat up at

9 months, stood alone and walked at 13 months and erupted his first teeth at 7 months of age. He fed himself fairly well; however, he was not toilet trained and had always been fussy and irritable.

He was the product of a fourth and full term pregnancy terminating in an uncomplicated ten hour spontaneous delivery at Freedman's Hospital. The mother had no illnesses during her pregnancy. His birth weight was eight pounds and there was no neonatal jaundice, anemia, convulsions or cyanosis.

He had had an occasional cold since birth and uncomplicated mumps and measles during the third year.

Routine immunizations were performed at approximately nine months of age. He had apparently received an adequate diet since birth.

His family history was essentially negative except that the father, who was 34 years of age, had a moderately severe case of Multiple Neurofibromatosis, apparently confined to the skin. Three other siblings were living and well. One sibling had died at Gallinger Municipal Hospital in 1945 at 14 months of age with "whooping cough and two convulsions." There was no family history of lues, tuberculosis, anemia or neurological disorders.

After admission to the ward the child slept for 5 hours and 45 minutes when he had another generalized clonic convulsion lasting about an hour which was controlled by 4½ grains of phenobarbital. Following this convulsion re-examination revealed essentially the same findings as were present on admission. Temperature at this time was 99.4°. For the remainder of the day the child slept and at 8 p.m., 17 hours after admission, he spiked a fever of 104° with a pulse rate of 170 per minute. Physical examination at this time revealed a comatose child with flaccid extremities, rapid cardiac rate and rapid even respirations. At this time the patient appeared moderately dehydrated with markedly inflamed tonsils and a mild right otitis media. With wet packs and intravenous fluids the fever was brought down to 102.6°. A spinal tap at the time revealed 10 mgm. % of protein, 45 mgm. % sugar and less than one cell. Carbon dioxide combining power following the intravenous injection of glucose and 1/6 M lactate was 49 vol. %.

During the night the child slept well and received subcutaneous fluids; however, his temperature remained around 103° and the following morning penicillin was begun. Physical examination at the initiation of the penicillin (30 hours after admission) revealed a temperature of 103° with a left hemiplegia, a left central facial paralysis, a left positive Babinski, stiff neck, hypertrophied inflamed tonsils and mild injection of the right ear drum. The eye grounds were normal. Blood pressure was 110/70. A repeat spinal tap at this time revealed less than one cell. This listless state persisted and on the 5th hospital day the temperature was normal and on the

7th day all signs of infection had subsided. The left hemiplegia was gradually becoming a left hemiparesis with minimal motion of the left upper and lower extremities. The eyes were deviation to the right and fixed and the left central facial paralysis was no longer present.

On the 8th hospital day penicillin was discontinued and on the 9th day the temperature again spiked to 102.6° and a diagnosis of pharyngitis and bronchopneumonia was made. An x-ray of the chest confirmed this diagnosis. Penicillin therapy was re-instituted and the temperature returned to normal by crisis. Neurological examination on the 9th hospital day revealed the additional positive finding of a left homonymous hemianopsia.

During the first 21 days of the hospital stay the child refused to eat and was maintained on clyses and gastric gavages. After this time he began to eat slowly, first liquids and later a regular diet. On discharge he was sitting in a chair with support and feeding himself fairly well. At the time of discharge his physical examination was entirely negative except for a mild left hemiparesis and left homonymous hemianopsia.

A pneumoencephalogram performed on the 14th hospital day revealed the ventricles to be well outlined and to be larger than seen in a child of this age. In the anterior-posterior view the anterior lateral horn on the left side was considerably enlarged as was the 3rd ventricle. On the posterior-anterior view, however, this was not seen (this type of picture is usually seen in normal post-convulsive ventricular systems). There was no evidence of increased intracranial pressure and the sella turcica was normal.

With the exception of a low grade fever for 3 days following the pneumoencephalogram the temperature was normal until discharge on the 33rd hospital day.

Additional significant laboratory findings are as follows: spinal fluid Kahn negative, blood standard Kahn and Mazzini negative, old tuberculin and Schick negative, blood counts and urinalyses essentially negative and no sickling of red blood cells in a 24 hour smear preparation.

DISCUSSION

The problem of hemiplegia developing some months after birth represents one which is not always satisfactorily explained. The case presented is one of these problems, but by the process of elimination probably falls into a group described as "acute infantile hemiplegia of vascular origin." A statement in Ford's "Disease of the Nervous System in Infancy, Childhood and Adolescence" seeming to fit this case is as follows: "In general, it seems correct to state that hemiplegia of apoplectic onset in a child may be regarded as presumptive evidence of a cerebral vascular lesion just as it is in adult life."

Many cases of acute infantile hemiplegia which have been considered to

be of obscure etiology prior to death have at postmortem been ascribed to vascular lesions involving the middle cerebral artery or vein. Other cases have revealed no demonstrable brain pathology at postmortem examination; however, later studies may bring out other etiological factors responsible for the syndrome.

Acute infantile hemiplegia of obscure or vascular etilogy has previously been described under the heading of "Marie Strumpell encephalitis" and "polioencephalitis"; however, postmortem examinations in most cases have failed to demonstrate the existence of an encephalitis and for that reason these terms have been discarded by most observers. Strumpell's belief is that these cases represent cerebral localizations of the virus of epidemic poliomyelitis; but this observation has not been satisfactorily proven.

The diagnosis of acute infantile hemiplegia of vascular origin is usually based on the elimination of other causes (which will be discussed further in this paper) and the clinical picture as described below. This type of hemiplegia is not rare and usually occurs before the age of 6–10 years in one who has been in apparent good health for months or years. The onset usually begins with unilateral or bilateral convulsions and coma, the convulsions recurring for several hours or days after which time consciousness is regained and the hemiplegia, hemianopia and other focal signs are noted. A temperature elevation up to 103° and frequent vomiting episodes usually accompany the convulsive period, following which the temperature returns to normal, barring complications.

Hemianopia, as occurred in this case, is usually considered unusual. The eyes are generally deviated to the side of the paralysis; however, they were deviated to the opposite side in the case presented. There may be a temporary aphasia if the lesion is on the left side of the brain.

The paralysis following the convulsion is at first complete, then a rapid recovery of function ensues with usual complete recovery of the facial paralysis, almost complete recovery of the leg paralysis, and less complete recovery of the arm paralysis. ,The arm usually remains permanently crippled due to the later development of a flexion contracture. A mild limp usually exists for the remainder of the patient's life. Other physical developmental deformities occurring several months after the development of the hemiplegia are small, underdeveloped distorted limbs and athetoses or tremors, the latter being rare. In other cases, as in the case presented here, the signs of hemiplegia may largely or completely disappear leaving no resultant spastic deformities. In a few instances the hemiparesis is a flaccid one.

The spinal fluid is negative in contradistinction to an encephalitis or poliomyelitis.

According to Ford,[1] convulsions recur from time to time in approximately

one-half of the cases. Paralyses may disappear shortly after the initial seizure only to return in a transient form after each additional seizure.

Most of the cases are mentally deficient and repeated seizures are thought to increase this mental retardation.

The prognosis is poor as true recoveries are extremely rare. Approximately 50% of the cases have recurrent convulsions and are mentally defective. The majority recover enough power to walk; however, the arm usually remains useless as mentioned previously.

Treatment in the acute stage consists of adequate control of seizures with sedatives and in the chronic stage of orthopedic care as well as sedative control of the convulsions.

Other causes of acute hemiplegia in infants and children to be eliminated before the diagnosis of acute infantile hemiplegia of vascular origin can be made will now be discussed.

Congenital hemiplegia, in most instances, is due to a cerebral birth injury, although rare instances are due to intrauterine injury or disease or to developmental defects. Hemiplegia is the most common type of paralysis resulting from a birth injury, the injury usually causing a cerebral hemorrhage or arterial thrombosis. This type of hemiplegia generally produces a picture similar to that described under acute infantile hemiplegia of vascular orgin, except that in congenital hemiplegia there is usually a history of birth trauma and the hemiplegia appears within a few months after birth. Facial pals, is also extremely rare. Developmental defects of the affected limbs are noticed early in life due to the early appearance of the hemiplegia. This type of hemiplegia may be confirmed in some cases by encephalography or ventriculography. Little clinical improvement is expected in a congenital hemiplegic, for a course of recurrent convulsive seizures followed by progressive mental deterioration is the rule.

Acute infectious diseases are occasionally followed by hemiplegia. The most common of these offenders are diphtheria, scarlet fever, influenza, pneumonia, typhoid fever, pertussis, measles, dysentery and typhus. The pathology here involved is usually the result of a single lesion, in contradistinction to so-called "hemorrhagic encephalitis," and for that reason there is usually no similarity to these two clinical entities. The lesions involved are either vascular (cerebral artery thrombosis and cerebral hemorrhage), embolic or infectious (abscess) in type. For example, diphtheria may cause a mural cardiac thrombosis with a resultant cerebral embolus, and pertussis a cerebral hemorrhage due to a persistent violent cough. In most instances hemorrhage is the initial process, later being followed by thrombosis on a toxic and degenerative basis. These cases are usually etiologically obvious due to their occurrence during the convalescent period of or several weeks after such an infectious process. The spinal fluid may be normal or there

may be as many as 2000 cells per cubic millimeter. Prognosis in cases such as these is very poor when hemorrhage is the etiological factor and most of these cases are fatal. Those due to embolism or thrombosis usually survive; however, they generally are crippled by mental and physical residuals. Treatment consists only of proper nursing care.

Also to be considered is a cerebral embolus and abscess resulting from a lung abscess, otitis media with mastoiditis, or bacterial endocarditis with septicemia. An uncomplicated hemiplegia following encephalitis is extremely rare, as most of these cases develop a Parkinsonian syndrome.

Neurosyphilis is very rare in infants and can readily be ruled out on negative serological tests for syphilis, negative long bone x-rays and absence of a clinical picture of lues.

Other diseases causing hemorrhage or thrombosis such as purpuras, scurvy, anemia, leukemia and hemophilia are to be considered.

Congenital heart disease with an embolus from a mural thrombus producing a middle cerebral artery occlusion must be remembered.

Also to be thought of are intoxications and disorders of metabolism (these usually produce diffuse processes), malignant hypertension and nephritis, lead poisoning encephalopathy, cerebral trauma (diffuse usually), middle cerebral artery aneurysm (rare), tetany and meningitis.

Vascular anomalies such as venous angiomas make an interesting differential diagnostic problem which is not always settled. They may differ from the case presented in that the hemiplegia may not appear until many years after the initial convulsive seizure. Increased intracranial pressure is rare. The diagnosis is rarely made prior to death in cases with uncomplicated hemiplegic symptoms, these cases generally falling into the category of the case here presented.

Arterio-venous fistulae usually produce signs and symptoms consistent with those of an extensive hemorrhage.

Degenerative processes such as Schilder's disease may produce an acute hemiplegia; however, these are extremely rare.

True neoplasms in this age group are also extremely rare and when hemiplegia results from one it is usually due to hemorrhage into the new growth or edema about it. The presence of a mild hemiplegia existing for over 2 years and negative pneumoencephalographic studies in the case presented tend to rule out a neoplasm.

Tuberous sclerosis and neurofibromatosis usually produce a diffuse type of cerebral involvement and paralyses are rare. Convulsive seizures generally are slow and progressive in development, usually beginning with focal twitchings without loss of consciousness. Later these seizures become more frequent and finally develop into violent generalized convulsions.

SUMMARY

1. Presented is a probable case of acute infantile hemiplegia of vascular origin.

2. The diagnosis is based mainly on the exclusion of all other possibilities.

3. A differential diagnosis is briefly discussed.

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HYDRONEPHROSIS: A CASE REPORT AND A REVIEW OF THIRTY-ONE CASES

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There are few conditions in medicine so frequently overlooked as the obstructive lesions of the urinary tract. Each delay in diagnosis allows irreparable damage to the kidneys. Any sign, symptom or laboratory finding suggestive of an anomaly or obstruction of the urinary tract should therefore be thoroughly studied as soon as possible. The following case report illustrates the history, physical examination, laboratory findings and manner of diagnosis of an obstructive lesion of the urinary tract.

R. W. 45-10326

R. W., a 23 month old colored male, was admitted to Children's Hospital on October 13, 1947 with complaints of dysuria, sore throat, cold, and diarrhea. The birth weight was 5 lbs. There was no history of neonatal convulsions or cyanosis. At six weeks of age, the patient was first seen in the dispensary for a mild upper respiratory infection and diarrhea. The weight was 8 lbs. 12 ounces, the temperature normal and the physical examination negative. The mother stated that the child at 10 weeks "strained a little." At 18 weeks of age, the patient was again seen in the dispensary presenting generalized edema. This was thought to be allergic in nature, and the patient was given a Mullsoy formula with improvement of symptoms. The patient gained weight satisfactorily on this regime but was seen intermittently complaining of alternating constipation and diarrhea. On August 15, 1946, at 9 months of age, the urine was reported as "loaded with white blood cells." (This was the first laboratory finding that suggested kidney pathology.) During the next three months the child was seen with repeated attacks of "naso-pharyngitis" with temperature of 101° to 101.8°. The mother reported on December 20, 1946 that the boy "cried when he makes water." The foreskin was retracted and a circumscision scheduled. However, the child continued to be seen in the dispensary with fever and symptoms of an upper respiratory infection. On one occasion the temperature was 104° with the chief complaint of "straining at stool." On each of these clinic visits, sulfadiazine was given with good results. From the winter of 1947 to midsummer 1947, the patient did not visit the dispensary. During July, August and September diarrhea, dysuria and intermittent fever were present again. Each time the examiner found a mild upper respiratory infection in a poorly nourished, chronically ill child. The urine on October 13, 1947 contained many white blood cells. The patient was

then admitted to the hospital to rule out a congenital urinary anomaly or cystic fibrosis of the pancreas.

Further information obtained from the mother at the time of admission included a history of frequency, cloudy urine, "milky drops" after voiding, polydipsia, melena, abdominal pain, occasional vomiting, anorexia, irritability and insomnia.

The family history was non-contributory except for a questionable history of syphilis in the mother 17 years ago. The physical examination revealed a well developed, poorly nourished colored male of two years in no acute distress. The head, eyes, ears, nose, throat and chest were not remarkable except for hypertrophic tonsils. The abdomen was slightly distended, but no masses or organs were palpable. There was no abdominal tenderness or rigidity, nor any costo-vertebral tenderness. Rectal examination revealed a smooth, moderately firm, non-tender mass thought to be a distended bladder.

Urinalyses showed from 0–150 mgm. of albumin, few to many white blood cells (clumped), rare to moderate number of red blood cells and occasional epithelial cells. The specific gravity ranged from 1.005–1.010. Hemogram showed 10.5 gm. of hemoglobin with 3,100,000 red blood cells, leucocytes varying from 4,700 to 14,000 with 61% polymorphonuclears. The urine culture was positive for E. coli and non-hemolytic Staphylococcus albus. The Kahn was reported as negative and the non-protein nitrogen blood level was 46 mgm. %. Pyelographic studies revealed bilateral hydronephrosis, bilateral hydroureters with enlarged bladder due probably to a posterior urethral neck obstruction. Cystoscopy later demonstrated a valve in the posterior urethra.

With the above case in mind, and with the thought that possibly something of value could be learned, the proven cases of hydronephrosis seen at Children's Hospital in the last five years were reviewed. The fact that some children were admitted in shock, some having convulsions, some with the diagnosis of ruptured spleen, diabetes insipidus, fecal impaction or pharyngitis, and some with the diagnosis of fever of undetermined origin is testimony that the symptoms may be extremely variable.

There were 31 cases of hydronephrosis at Children's Hospital during the past 5 years. Of these, 20 were males and 11 females. There were 23 white patients and 8 colored. The average age at the time of admission was 2 years $10\frac{1}{2}$ months, the youngest being 20 days old and the oldest 12 years. The time of onset of symptoms was known in 28 cases and undetermined in 3. The age of onset was under 2 years in 20 cases (71.4%). Four patients (14.2%) began having symptoms between 2 and 5 years of age, and 4 (14.2%) began after 5 years of age. The average age at onset of symptoms was 1 year 10 months. Thus, an average of 1 year elapsed from onset

of symptoms to diagnosis. Six cases (19.35%) are known to be dead, but this figure may be higher as follow-ups were not possible on all cases.

The prominent symptoms that these patients presented are significant. Twelve (38.7%) had definite urinary complaints, 10 (32.2%) had complaints that could be urinary or otherwise, but were not definite, and 9 (29%) showed symptoms ordinarily unrelated to the urinary tract. The most common symptom was fever. Fourteen (14) of the 31 patients had fever, either alone or associated with intestinal or urinary symptoms. The next most common symptom was vomiting, 10 showing this manifestation. Nine patients exhibited dysuria, eight had abdominal pain and seven showed polyuria. The other complaints in order of frequency were oliguria, hematuria, pyuria and listlessness, polydipsia, diarrhea, anorexia and incontinence, convulsions and constipation. Of the first four most common symptoms, only dysuria (9 of the 31 cases) was definitely a urinary complaint. This brief tabulation reveals the relative infrequency of definite urinary manifestations in patients with hydronephrosis.

The recorded findings of the examining physician are revealing. Fourteen (45.1%) showed essentially negative physical examinations. Of the remaining 17 with positive physical findings, 13 (76.5%) had an abdominal mass. This significant figure shows that the diagnosis is not made until late in the disease, or that symptoms of this condition are not present until marked damage has occurred. Ladd and Gross[1] state that "while a kidney pelvis can attain enormous proportions and even fill half of the abdomen, it may be unaccompanied by pain in its dilated state." This is more reason why this condition must be thought of early and often. Other significant findings include rectal mass (2), abdominal tenderness (1), abdominal distention (2) and imperforate anus (2). Two patients were recorded as poorly developed and poorly nourished; two were admitted in shock, and one was admitted convulsing. It is interesting to note that not one admission physical showed any costo-vertebral angle tenderness. This can be explained by the difficulty one has in eliciting lumbar tenderness in an infant or child, or by the fact that the examiner, not suspecting kidney involvement, failed to determine if kidney tenderness was present or not. The textbooks do not mention costo-vertebral tenderness as a prominent finding.

Of great help in bringing the physician's attention to the urinary tract was the laboratory. The simple procedure of a complete urinalysis, too frequently overlooked, was of utmost importance. Twenty-seven had definite abnormal urinary findings, 3 had no urine reports and one patient showed a normal urine. Twenty-three of the 27 abnormal urines contained pus cells, 17 showed albumin and 7 contained erythrocytes. The hemograms showed 6 patients with less than 10 gm. of hemoglobin and 14 with a

leucocyte count of more than 10,000. Of 15 non-protein nitrogen studies, 12 were above 35 mgm. %. Fifteen positive urine cultures were reported, but cultures were not taken on all patients, and on some only after treatment (sulfadiazine, etc.) was instituted. The organism most frequently found was E. coli (10 times). Non-hemolytic Staphylococcus was cultured from 4 urines; hemolytic staphylococcus was cultured from 4; non-hemolytic streptococcus from 2; and A. aerogenosa from one.

Roentgenological studies play the most important rôle in establishing the definite diagnosis of hydronephrosis. One patient was diagnosed previous to being sent to the hospital for nephrectomy. Four cases were discovered at autopsy, the correct diagnosis not even being suspected in three of these. There were 27 pyelographic studies done, 26 showing positive evidence of hydronephrosis. In addition to the hydronephrosis, 9 cases showed other urinary tract abnormalities. There were 6 non-functioning kidneys. One of these was found to have a hydronephrosis at autopsy; 2 were shown to represent agenesis of the kidney (also by autopsy) and 3 were not determined. There were 2 reduplications of the ureters with a supernumerary kidney in one case, and one bifid pelvis. In addition to x-ray evidence of kidney anomalies, one case was thought to be due to a polycystic remnant, and another case was due to an ectopic kidney. Thus eleven cases showed some congenital malformation of the urinary tract.

It is not sufficient for the diagnosis of hydronephrosis and hydroureter to be made without determining the point of obstruction. In this respect, cystoscopy is of utmost value. Advances in pediatric urological diagnostic procedures and treatment offer greater opportunities for correct diagnosis and effective treatment. In this series, the etiology could not be determined by x-ray, cystoscopy or autopsy in 16 cases (51.6%). Four of these had other congenital anomalies. Of the remaining 15, four had posterior urethral or bladder-neck obstruction; four had uretero-pelvic obstruction, two of which were due to aberrant renal vessels; three had uretero-vesical obstruction. Extra peritoneal pelvic masses caused obstruction in two cases, and an ectopic (pelvic) kidney with kinking of the ureter was the pathogenesis of one case.

This is a small series of cases but the conclusions that can be drawn are similar to more extensive studies. The fact that obstructive lesions of the urinary tract occur producing varied signs and symptoms which delay the correct diagnosis is demonstrated.

It was shown that about one year elapsed between the average age of onset of symptoms and the average age of diagnosis. Kretschmer[2] stresses the importance of early diagnosis so that early treatment can be instituted to prevent kidney damage.

Recurrent attacks of fever with abnormal urinary findings which fail to

clear on ordinary methods should lead one to pyelographic and cystoscopic studies which in almost every case will show the true diagnosis. It would be inadvisable to recommend pyelographic studies in all cases with only one attack of pyelitis or pyelonephritis. However, the possibility of hydronephrosis should be kept in mind, and subsequent attacks, especially in the male, warrant a complete urological examination. Hydronephrosis may be suspected clinically, but the radiologist and urologist make the definite diagnosis.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.

Assisted by: Harold W. Bischoff, M.D.

Adrian Recinos, Jr., M.D.

By Invitation: George William Ware, M.D.

Harold W. Bischoff, M.D.

A three and one-half year old white boy was brought to Children's Hospital on November 28 because of abdominal pain of two days duration. He had had a cold for three days and on the night before the onset he was given a laxative which was "not very effective." The pain was in the periumbilical region at first and was inconstant. On the day of admission it had shifted to the right lower quadrant. He vomited twice and received an enema which yielded poor results.

He had had occasional colds and the measles but there had been no other illnesses. The parents were well and this was their only child.

The boy was flushed but comfortable and his temperature was 103.4°. His throat was injected and there was a nasal discharge and a few palpable cervical lymph nodes. Respirations were slow and regular. The abdomen was slightly rigid on the right side. There was generalized tenderness which was most marked in the right lower quadrant. A routine urinalysis was negative and the red blood cell count was 3.38 million with 10 grams of hemoglobin. The white cell count was 16,000 with 71% polymorphonuclear forms, 8% band forms, 10% eosinophiles and 11% lymphocytes. A tuberculin test, examination of the stool for ova and parasites, chest x-ray and a barium enema were all negative.

After the second day the temperature remained essentially normal. He felt better but tenderness persisted on the right side of the abdomen. On the eighth day a firm, walnut-sized mass was felt on the right by rectal examination. This enlarged somewhat and then resolved in two weeks during which the white cell count varied from 9,400 to 12,200. Two more urinalyses were negative and the stools remained normal. After twenty-three days of hospitalization he was symptom free and was discharged.

Three and one half months later he was readmitted because of pain and vomiting of two days duration and inability to urinate of twelve hours duration. Since the previous admission his health had been good until diarrhea and vomiting began one week before readmission. His appetite for solid foods was poor though he took fluids avidly. He complained of pain in the lower abdomen and pain on urinating. During the 12 hours prior to ad-

mission he was unable to urinate and was dribbling slightly. Shortly after entry to the hospital he voided about 400 cu. cm. involuntarily.

Nourishment and development were good. The abdomen was soft but there was tenderness over both lumbar regions and tubular masses were felt in both flanks. An intravenous pyelogram suggested a bifid pelvis on the left with mild hydronephrosis and marked hydroureters. The specific gravity of the urine was 1.020 and there were a moderate number of white and red cells in the sediment. One urine culture was sterile but six other specimens were cultured and Staphylococcus albus was obtained from all of them, non-hemolytic streptococci in three of the specimens and E. coli once in various combinations. The red-cell count was 4.04 million and white count was 20,800 with 50% polymorphonuclears, 24% band forms, 4% metomyelocytes, 3% unclassified cells and 19% lymphocytes. The non-protein nitrogen was 26 mgms. %.

During the first week sulfathiazole was administered and the temperature was sustained at 102°. Thereafter until operation it was normal. Low abdominal pain persisted but the other complaints regressed. Because a posterior urethral valve was suspected a cystoscopy was performed. No anomaly was found but the bladder was distorted by a retrovesicular mass which rectal examination under ether anesthesia revealed to be firm, knotty and irregularly shaped. It was between the rectum and the bladder and extended three quarters of the way to the umbilicus. A cystogram and x-ray examination of the chest and spine were normal.

An operation was performed on the 11th day of hospitalization.

DISCUSSION

George W. Ware, M.D.: On the first admission to the hospital the most likely diagnoses were (1) appendicitis and (2) upper respiratory infection with associated mesenteric adenitis. We would like to discuss these before proceeding to less likely diagnoses.

Appendicitis is the first diagnosis which comes to mind and rightly so in the case of abdominal pain in a child. The diagnosis is suggested by abdominal pain which shifted to the right lower quadrant, by vomiting, and by right sided rigidity and generalized tenderness most marked in the right lower quadrant. Fully as important as any of these symptoms is the fact that the night before onset of the pain, he was given a laxative. It is a well known fact that the ingestion of a laxative will confuse the physical findings of appendicitis. Hence with such a history the surgeon is always more suspicious.

The combination of a cold, abdominal pain which was periumbilical, vomiting, flushed facies, temperature of 103°, injected pharynx, nasal discharge and cervical glands were all consistent with a diagnosis of mesenteric adenitis.

These two are the most likely diagnoses considering the history, physical findings and routine laboratory work. It is unusual in either condition to have the temperature drop to normal in a short time and remain so. However, in my opinion the patient was forming an appendiceal abscess and with the walling-off process, toxicity abated. This is substantiated by the latency of events especially by the mass felt by rectum. This mass also, to my mind, rules out mesenteric adenitis. Hence it is a good possibility that this patient had acute appendicitis with subsequent abscess formation on his first admission to the hospital.

Other diagnoses to be considered are kidney infection, polycystic kidney with hemorrhage, allergic enteritis, worm infestation, pneumonia and rheumatic fever. We will take each of these up in turn.

A kidney infection would have to be considered on the basis of history and physical findings. This diagnosis is compatible with a cold for several days, vomiting, abdominal pain and tenderness. However, it is made less tenable in view of successive negative urinalyses, normal temperature and rectal mass.

It is a well known fact that hemorrhage into a polycystic kidney might cause the signs of a surgical abdomen. This would also account for the elevation of temperature with subsequent drop to normal and localized pain considering a unilateral hemorrhage and leucocytosis. Urinalyses may be normal since there may be no connection between the area of hemorrhage and the pelvis of the kidney. If such a mass could be felt by rectum it would certainly be felt abdominally and no mention is made of an abdominal mass. A urinalysis showing good specific gravity is also against such a diagnosis.

The eosinophilia noticed on the first white blood count raises the question of allergic enteritis. Such a condition would cause abdominal pain, vomiting, slight rigidity of the abdomen, generalized tenderness and flushed facies. However, there is no history of a preceding allergy which one might expect in a three and one half year old boy. There is no mention of headache which is a common symptom and similarly there is no diarrhea which one would expect with such a condition.

Worm infestation is also mentioned because of the eosinophilia. It would be extremely unusual for an uncomplicated worm infestation to run such a course. The only suggestion of its presence is the eosinophilia. Blockage of the appendiceal lumen by pin worms is a proven pathological fact. The symptoms and signs which result from such a condition are those of appendicitis and not those of pin worm manifestation.

Pneumonia is considered briefly because of the preceding upper respiratory infection and associated abdominal pain. Although pneumonia may be difficult to rule out in some cases of abdominal pain I do not believe this to be the case here. Against such a diagnosis is a statement that the

patient was comfortable and that his respirations were slow and regular. There is no mention of physical findings in the lungs; however, in the presence of a negative chest film we can assume that they were normal.

Rheumatic fever is mentioned only to emphasize its importance in the differential diagnosis of a possible surgical abdomen in a child. There is nothing to suggest rheumatic fever in this patient. Against such a diagnosis is his age, fall in temperature, lack of joint symptoms, absence of cardiac findings and a negative chest film. When rheumatic fever does cause abdominal pain and tenderness they are not usually as localized as in this case.

The second admission immediately focuses our attention on the genito-urinary system; however, considering his previous admission and attempting to make one diagnosis of all his complaints, we will attempt to correlate the two admissions. In regard to the genito-urinary tract, infection is the first condition which comes to mind. This is suggested by vomiting, abdominal pain, lack of appetite, dysuria, a soft abdomen and tenderness in the lumbar areas.

The second genito-urinary condition brought to mind is that of a polycystic kidney. It would explain lumbar tenderness and tubular masses in the flanks. Superimposed infection would explain the symptoms on entering the hospital. However, polycystic kidneys with or without infection are ruled out by the intravenous pyelogram and by the normal specific gravity of the urine, sterile cultures and normal non-protein nitrogen.

In attempting to explain the retro-vesicular mass the most probable kidney condition would be an ectopic kidney or more specifically a pelvic kidney. This possibility is immediately ruled out by the normal position of both kidneys on intravenous pyelogram.

Considering again his first admission and a diagnosis of appendiceal abscess, the possibility of the rôle of an inflammatory mass in the second illness comes to mind. It is known that appendicitis may go on to abscess formation and at a later date be the site of a severe infection for a second or a third time. Reinfection occurring in an appendiceal mass could explain pain, vomiting and diarrhea. Retention with involuntary dribbling and the pain on urination could be due to the pressure of an inflammatory mass on the bladder. This latter consideration seems more likely in view of the negative urinary findings from the laboratory standpoint. An appendiceal abscess pressing on the neck of the bladder could cause a bilateral hydroureter in the female seen in approximately the fifth month of pregnancy. In both instances the renal and ureteral pathology is caused by extrinsic pressure on the bladder. This possibility is supported by cystoscopic findings which revealed the bladder to be distorted by a retro-vesicular mass which could be felt between the rectum and the bladder.

Such a diagnosis, however, does not explain a soft abdomen, bilateral lumbar tenderness and bilateral tubular masses. I am very concerned with the report of these tubular masses. It has been suggested that these masses might well be the ascending and descending colon. I accept this suggestion because I myself am unable to explain them.

A central nervous system lesion might be considered because of the overflow incontinence. However, there is no history of trauma nor is there any statement regarding abnormal neurological findings. There is no mention of retention or involuntary voiding following admission. Considering these facts I feel the diagnosis of central nervous system lesion can be safely discarded.

The possibility of a malignancy must be considered. Considering the age group, a Wilm's tumor or neuroblastoma comes to mind. A malignancy would certainly explain the mass behind the bladder and a subsequent hydronephrosis. A mass behind the bladder and not felt elsewhere would militate against the diagnosis of Wilm's tumor or neuroblastoma. Also against the diagnosis of malignancy are the long duration of the illness, the good nourishment and development, the absence of liver enlargement, negative x-rays of the chest and spine and normal intravenous pyelogram. The extensive size of the mass certainly suggests that if it were a malignancy numerous metastases would be present.

In summary I would consider the most likely diagnosis to be an appendiceal abscess.

PATHOLOGICAL DISCUSSION

E. Clarence Rice, M.D.: Dr. Ware's diagnosis is correct. At the time of operation the impression was that the child might well have an abdominal malignancy. On his previous admission the possibility of either appendicitis or mesenteric adenitis was considered, the latter being the final diagnosis at the time of his first discharge from the hospital.

The continued illness could not be explained on the basis of a respiratory infection which the patient had initially. The bouts of fever, anemia and leucocytosis would certainly make one suspect the presence of an infection. The urine cultures would tend to support such a finding; however, the microscopic examinations of the urine sediments prior to operation were not in keeping with the diagnosis of a urinary tract infection. A perinephritic abscess is often due to a staphylococcus infection. I would suspect that a number of the cultures were contaminated.

Dr. Ware mentioned rheumatic fever as a cause of abdominal pain. I would remind you of two other conditions which a surgeon has to keep in mind with reference to abdominal pain, viz. diabetic acidosis and the crisis of sickle cell anemia. However, these are seen in acute illnesses, not chronic ones.

Eosinophilia did not appear until the second admission and may have been due to a bacterial sensitization as there was no other evidence of allergy or parasitism. The commonest abdominal malignancies in children of this age are adenomyosarcoma (Wilm's tumor), neuroblastoma and lymphosarcoma. The presence of metastases would have undoubtedly made themselves evident during the five months that the patient was under observation had the last two named diseases been present. Wilm's tumor would be more likely to be confused with the observations noted in this patient; however, he was not emaciated or cachectic at the time of his second admission which was four months after his initial discharge.

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